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CHRONIC ATROPHIC RHINITIS --

ITS PATHOLOGY AND AETIOLOGY,

by

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There are many terms used as synonyms for this disease:- Chronic Atrophic Catarrh, Chronic Foetid Rhinitis, Coryza Foetida, Ozaena, French - Ozène, German - Stinknase.

The name Chronic Atrophic Rhinitis appears to give the best description. The essential feature of the disease is a slow and progressive atrophy. The term Chronic Atrophic Catarrh is not to be objected to, but that of Chronic Foetid Rhinitis merely indicates a long lasting disease of the nose accompanied by foul odour. Coryza Fetida means a foul-smelling cold in the nose, and Ozaena means still less -- being only the Greek for stench.

Foul-smelling conditions occur in the nasal passages quite apart from the disease under consideration, as, for example, in syphilitic ulceration and necrosis, glanders, foreign bodies, malignant ulceration, chronic suppuration of the sinuses, etc.

Any term describing the disease as a stench would be erroneous, because it sometimes occurs unaccompanied by foetor. The stench which is usually present would seem to be an accompaniment, but not an essential part, of the disease. It is the most distressing feature, and the one which brings these cases under the phy-

sician's notice. Probably there are many cases of Chronic Atrophic Rhinitis which have never been treated, on account of those who are afflicted having no ozaena accompanying the condition.

The term Chronic Atrophic Rhinitis is now almost universally used by rhinologists -- vide Zuckerkandl - Anatomie der Nasenhöle, Vol. II; Tilley - Diseases of the Nose and Throat, 1908; Hartmann -- Atlas der Normalen und Pathologischen Anatomie der Nase, 1891; Herbert F. Waterhouse - Quain's Dictionary of Medicine, 1902; Charles A. Parker - The Nose and Throat and their Treatment, 1906; E. B. Wagget - Diseases of the Nose, 1907. It is to be noted that collapse of the vascular structures of the nasal cavity, accompanied by deficient secretion, is called by some Atrophic Rhinitis; this must not be confused with the disease under discussion, in which we have true atrophic changes which are absent in the so called Atrophic Rhinitis above-mentioned.

The accompanying photograph (No. 1) is taken from a typical specimen of the disease which I procured while acting as demonstrator of Anatomy at the Royal College of Surgeons, Edinburgh. The subject was a male, aged about 60, who was killed by an accident. Unfortunately I was unable to get any details of his history during life. The body was well nourished and strongly built. It shewed no appearances of chronic

disease other than that found in the nose. His arteries, etc., were in a healthy state. On looking at the parts in the proximity of the nose, I found the mouth was in a healthy condition, with the exception that the teeth of the upper jaw were absent. The tympanum shewed to the naked eye no abnormality.

The cadaver was in a fresh state when I first examined it, which excludes any marked changes in the soft parts, which might have occurred, due to evaporation of fluids or preparation for dissection.

In the photograph it is seen that the specimen has been cut in sagittal section. I removed the septum, which ^a~~was~~ attached to the left half of the specimen, in order to expose the left outer nasal wall.

The septum shewed no abnormal deflection, no spines, and no perforation or ulceration.

The septal mucous membrane shewed the same atrophic changes as those found in the mucous membrane covering the nasal cavity in general.

The morbid anatomy of this specimen is very marked, and very characteristic of an advanced stage of the disease. I will now proceed to describe its naked eye and microscopical appearances as compared with those found in the normal condition. I will also endeavour to bring out some points of interest which are not dealt with in the various monographs on the subject.

A comparison of the measurements found in this

of
 pathological specimen with those in an average normal
 nasal cavity brings out more clearly the altered con-
 ditions presented by the disease. I have not been
 able to find that any such measurements have been made
 before, and I venture to think they will aid greatly in
 the appreciation of the condition.

In Quain's Anatomy, 1894, Vol. III, Part III, p. 134,
 the following measurements of the normal nasal fossae
 are given (Thane):-

- I. Greatest vertical measurement (at fore part of
 cribriform plate) - - - - - 44 mm.
- II. Greatest sagittal measurement along floor (from
 posterior extremity of hard palate to anterior
 extremity of roof) - - - - - 73 mm.
- III. Sagittal measurement of osseous part of
 floor - - - - - 44 mm.
- IV. Least sagittal measurement (close below
 cribriform plate) - - - - - 35 mm.
- V. Greatest coronal measurement (near floor) - - 16 mm.
- VI. Least coronal measurement (near roof) - - - 2.5 mm.

In examining several normal nasal cavities, on the
 above lines and on lines of my own, I find they vary
 greatly, but none approach the peculiar variations
 present in my specimen of Chronic Atrophic Rhinitis.
 Antero-posteriorly the whole cavity is abnormally short,
 and transversely it is abnormally wide. There is a
 peculiar guttering of the nasal floor, which I have

never seen described; and which must account for much of the increase of space of the air passage.

In examining the condition more systematically, I found nothing abnormal on the facial aspect. The orbits were not abnormally wide apart - their centres being 5.8 cm. from each other. The nose was short, but normal in appearance. The anterior nares were an average size; and looked in the normal downward direction. The bridge shewed a well rounded arch from side to side, and was of the straight variety from above downwards. The teeth of the upper jaw were absent, but the gums appeared healthy. There were no signs of disease in the mouth, tonsils, palate, oral pharynx, or laryngeal pharynx. In the naso-pharynx the eustachian cushions appeared somewhat smaller than usual, but the tubal orifices were not abnormally wide. The mucous membrane of the naso-pharynx was slightly atrophic, but not so markedly atrophied as that found in the nasal fossae. The morbid condition seemed to lie almost entirely within the nasal cavity. The measurements of this nasal cavity as compared with Quain's normal table, I have arranged as follows:-

Specimen of Chronic
Atrophic Rhinitis.

Quain's Normal
Measurements.

I. Greatest vertical measurement (at fore part of cribriform plate)	48 mm.	44 mm.
II. Greatest sagittal measurement along floor (from posterior margin of hard palate to anterior extremity of roof)	53 mm.	73 mm.
III. Sagittal measurement of osseous part of floor	37 mm.	44 mm.
IV. Least sagittal measurement (close below cribriform plate)	42 mm.	35 mm.
V. Greatest coronal measurement (near floor)	19 mm.	16 mm.
VI. Least coronal measurement (near roof)	7 mm.	2.5 mm.

The above table shews the marked difference between the measurements of the diseased condition and those of the normal.

It is convenient to mention here that I have measured several normal palates, and find quite a difference between Quain's measurement and my own. My average works out at 50 mm., as compared with Quain's 44 mm., for the sagittal measurement.

Turning to the Morbid Anatomy, the palate shews peculiarities not only in its being short but in its being very thin. Its centre is so thin that daylight shines through it with ease -- it is only 2 mm. in thickness, as compared with 3 mm. to 3.5 mm., which

is normal.

The palate process of the superior maxilla normally shews two distinct tables with cancellous bone between; here the palate process is one solid plate of bone.

The alveolar process of the superior maxilla is atrophied, due to the teeth being absent.

There is a peculiar gutter-like concavity in the floor of the nose on each side of the septum. This makes the floor of the inferior meatus concave from side to side. This seems to have been due to an atrophy of the nasal surface of the palate, in a downward and outward direction.

The accompanying diagrams (Nos. 2 and 3) shew the normal gentle curve taken by the outer nasal wall just before it joins the floor. This curve is seen to pass in a downward and inward direction, to become insidiously continuous with the floor.

Diagram No. 4 shews the condition of the floor and lower part of the outer nasal wall in the atrophic specimen. This guttering must increase the air space of the inferior meatuses to a great extent.

The posterior nares or choanae were of an average size, although the ease with which air passed through them must have been greatly increased, as the posterior ends of the inferior turbinates are completely absent, and the posterior ends of the middle shew marked atrophy.

In addition to these facts, the mucous membrane as it passed from the nasal cavity through the

choanae was thin and tightly stretched over these openings and must have been less encroached on by turgescence than in the normal condition.

The outer nasal walls shew striking features of the disease, the two sides being about equally affected, so that a description of one side - viz., the right side - will serve for both.

The inferior turbinal is practically absent: what remains of it is represented by a semilunar fold with its concavity looking downwards. This semilunar fold bridges over the nasal duct, which opens under cover of its anterior third. (See photographs Nos. 1, 2 and 3.)

The orifice of the nasal duct is widely patent; its margin is sharply defined. The diameter of its orifice is 1.5 mm.; it faces almost directly backwards, and it has no valvular fold of mucous membrane to guard it (the valve of Hasner).

On looking more closely at this semilunar fold -- at its anterior and posterior ends it is seen to become gradually blended with the outer wall. It projects only very slightly into the nasal fossa. The diagrams Nos. 3 and 4 shew the marked projection of a normal turbinal as compared with this semilunar fold whose septal surface is almost on the same plane as the external nasal wall. Its inferior or free border is very short, being only 23 mm. as contrasted with the length of the free border of a normal inferior turbinal, which is from 40 mm. to 50 mm. The thickest part of its free border is at the anterior extremity, and is only

2.5 mm. as compared with more than double that figure in the normal turbinal. The semilunar fold consists of a thin layer of bone covered with atrophic mucous membrane.

Passing to the middle turbinal, this bone is seen to present extensive atrophic changes — compare photograph No.1 with photograph No.3. It does not reach sufficiently far down to conceal from view the uncinate process and the region of the hiatus semilunaris. It does not reach far enough forward to conceal from view the opening of the infundibulum. The body and free edge of the bone are very thin, the former being only 2 mm. at its thickest part.

The line of attachment of the middle turbinal is normally placed, but less extensive than usual, being 26 mm. as compared with an average normal 38 mm. The atrophy appears to have taken place in great part from the free margin, although the whole bone is extensively atrophied, so that less bone projects into the nasal fossa. There is no pneumatic cell at its tip. (A pneumatic cell is present in 30% of normal middle turbinals - L.Turner.)

There is no bulla ethmoidalis present. There is but a feebly curved ridge representing the uncinate process. The hiatus semilunaris practically does not exist, because the uncinate process does not project sufficiently to form it.

The orifice of the maxillary antrum is normally placed, but widely patent, due to the atrophic nature of the mucous membrane.

The orifices of the ethmoidal air cells and the orifice of the infundibulum shew the same wide open appearance, their margins being thin and sharply defined.

In passing still higher, the superior turbinal is represented by a sharp edge of mucous membrane, which arches immediately above the orifice of the posterior ethmoidal air cells.

The arc represented by the third of the circumference of a threepenny bit would almost accurately represent this arch-like fold. (See photograph No.1.)

Above this, in the region of the spheno-ethmoidal recess, is the opening of the sphenoidal air sinus. This presents nothing abnormal, with the exception that it is covered with atrophic mucous membrane, similar to that found throughout the nasal cavity.

On looking through the anterior nares with reflected light, it is interesting to note that one could see the opening of the sphenoidal air sinus, and one could see past the inferior and middle turbinals into the naso-pharynx. This peculiarity was due to the insufficient projection of the turbinals causing no obstruction to the view. This is frequently noted in cases of Chronic Atrophic Rhinitis, but it is seldom so marked as in the case in question -- even after allowing for some post-mortem shrinkage of the soft parts. It is interesting to note, that the accessory sinuses shew nothing to indicate that the disease has reached them.

I opened the maxillary antrum from its facial surface, and inspected it from this aspect. It was

rather small in size, but not abnormally small. The mucous membrane was apparently quite healthy. There was but one ostium situated close to its roof. The cavity contained no débris, and there were no crusts or secretion sticking to the mucous membrane.

The other air sinuses were in the same apparently healthy state.

I made microscopic sections of their lining membranes, but could find no pathological changes present even in the more superficial cells.

If we now turn to the more minute changes presented by the atrophic mucosa, and study microscopic sections taken from the various regions of the nasal cavity, we find the pathological processes are most vividly shewn in the mucous membrane covering the inferior and middle turbinals.

Regarding the turbinals as organs associated with the respiratory function (especially the inferior turbinals), it will be understood that changes in their histological structure are of special moment.

The inspired air is warmed by passing over the extensive surface of mucous membrane which is presented by the normal turbinals; in virtue of the mucus secreted by the glands in the mucous membrane, the inspired air is also rendered humid before reaching the more distant air passages. The large cavernous spaces of the inferior turbinals, filled as they are with blood, add to the power which these organs possess

of warming the inspiratory air current.

The middle turbinals differ somewhat in structure from the inferior turbinals; they possess less cavernous tissue, and hence their erectile properties are less marked. The middle turbinals, however, ^{are} especially rich in racemose glands, which open by large orifices on to their surfaces. The deeper layers of the mucous membrane covering the middle turbinal are much more adherent to the bone than are those of the inferior turbinal -- thus forming a muco-periosteum for the bone. Microscopic sections cut from the specimen of Atrophic Rhinitis in question shew clearly the important changes seen in an advanced stage of the disease.

If the micro-photograph No.9 of the normal middle turbinal be compared with the micro-photograph No.8 of the atrophic middle turbinal, some very important changes are at once recognised.

The normal columnar ciliated epithelium has entirely disappeared, and all the surface folds and corrugations have become flattened out, so that the surface is now smooth and covered by stratified epithelium, which shews its more superficial layers to be degenerated and scaling off in places. Granules of pigment are seen scattered amongst the cells near the surface; these are probably the result of nuclear degeneration. The racemose glands, which are well shewn in photograph No.9, are here seen to have completely disappeared. Here and there, under the epithelial surface, are seen

open spaces in the connective tissue -- these correspond to areas which were originally occupied by racemose glands. The faint outline of alveoli lined by granular degenerated cells can be seen under the microscope, but not in the photograph.

There is an increase of connective tissue throughout the mucous membrane.

Of the cavernous spaces which remain, the majority are in a collapsed condition; the blood vessels are reduced in number, but their walls are not markedly thickened. The involuntary muscle has all disappeared. These latter changes are better seen in sections taken from the inferior turbinal.

The atrophy of the glands was so complete that there was no chance of finding fatty changes in the cells, as described by Krause and Hebermann (Tilley - Diseases of the Nose and Throat, 1908. McBride - Diseases of the Throat, Nose, and Ear, 1900).

The bone of the inferior turbinal shewed no histological changes, although of course markedly reduced in size. The surface of the middle turbinated bone shews little excavations at various points on its surface. In the region of these bays or depressions the connective tissue is loose in structure and occupied by cells with large nuclei. It is well seen in photograph No. 8. This was seen only in the bone of the middle turbinal, and is probably due to a secondary infection of the muco-periosteum. If these changes are compared with those found by Wingrave (see Tilley,

p.34), it is seen that they are somewhat similar.

Wingrave found:-

- "(1) the ciliated columnar Epithelium replaced by stratified Epithelium.
- (2) Diminution and degeneration of the glands and venous sinuses.
- (3) Beneath the surface Epithelium a stratum of round cells, more particularly located in the neighbourhood of the vessels and glands." (This I have seen in earlier cases of Chronic Atrophic Rhinitis; in my specimen a more fully formed fibrous tissue has taken the place of these cells.) "Amongst these cells are certain highly refractive bodies of unknown nature." (These I have not been able to find.)
- (4) "The bone is passively atrophied and shews no evidence of active disease." Further he mentions that "the brunt of the disease seems to fall on the glandular tissue."

These microscopical changes account for the decreased secretion of mucus and the loss of the vascularity of the whole mucous membrane.

Aetiology of Chronic Atrophic Rhinitis.

I look upon the change in the bone as the first stepping-stone towards the true appreciation of the Aetiology, because it is one of the most obvious and essential features of the disease when it is somewhat advanced.

Attacking the aetiology from this point of view, I may say, I do not believe in the theory that the bone is arrested in development, and that the disease is caused by this arrest in development. I believe arrest in development of bone plays no part in this disease, and this I will endeavour to prove.

It seems to me many authorities confuse the meaning of "Arrest in development" with that of "Atrophy." To make this clear, Coates in his text-book of Pathology, 1903, defines Simple Atrophy as diminution in the nutritive activity of structures, and a consequent diminution in size, without further change. He distinguishes this from Hypoplasia or Aplasia, which is a smallness due to defective growth. This latter is what I understand by the term "Arrest in development."

Seeing that the inferior turbinals shew the morbid changes most markedly and most constantly, a consideration of their development will make clear much of their Pathology. According to Morris, in his text-book of Human Anatomy, 1907, p. 74, the inferior turbinal may be regarded as a dismemberment of the lateral mass of the ethmoid. It is ossified in cartilage from a single nucleus, which appears in the fifth month of intra-uterine life. At birth it is a relatively large bone, and fills up the lower part of the nasal fossa.

The size of this bone at birth seems of great importance in bearing out the views here put forward - that the disease is the result of Atrophy, and not caused by arrest in development of bone.

Photograph No.4 shews the turbinated bodies in the outer nasal wall of a full-term foetus.

If the inferior turbinal in this specimen is compared with the adult bone (see photographs Nos.5 and 6),it will be seen to be similar in all detail,except that it is smaller in size. It,however,occupies the same relative position in ^{the} infantile fossa as the adult bone does in the fully developed fossa. If it is now compared with the remains of the inferior turbinal in the Atrophic specimen (see photographs Nos.1 and 2),it is impossible to believe that the latter condition could have resulted from an arrest in development in the infantile turbinal dating from birth. Because,where this ~~is~~ the case,surely the inferior turbinal in the atrophic specimen would be similar in size and shape to that found in the infantile nose. At a glance this is seen not to be the case,as there is practically no inferior turbinal in the atrophic specimen.

If we regard the condition as an intra-uterine arrest in development -- that is,an arrest in development before the bone reaches the dimensions present at birth -- what evidence have we that children are born with this condition?

In going over the literature on this subject,I found only one case of absence of the inferior turbinals,recorded by Von Hyrtl ("Congenital Malformations", Hektoen and Riesman,Pathology,1901). In cyclopic

monsters, etc., the nose may be entirely absent, but under these circumstances the individuals do not live.

In examining infants for nasal abnormalities at the Rotunda Hospital, Dublin; in my experience of the children I saw while working at the Nose, Throat and Ear department of the Royal Infirmary, Edinburgh, and the Throat, Ear, and Eye Infirmary, Cambridge St., Edinburgh; and in my experience of the foetal bodies and skulls which I examined while acting as Demonstrator of Anatomy at the Royal College of Surgeons, Edinburgh, I never came across a case of congenital absence of the turbinals, or even of congenital smallness of these bones. Were the absence of these bones a common occurrence, it would have been more frequently recorded. I have questioned authorities on this subject, but I have failed to find a record of another case.

From my work on this subject I conclude that intra-uterine arrest in development is of no value as an aetiological factor in producing this disease, because its occurrence is extremely rare, and, further, Chronic Atrophic Rhinitis is by no means a rare disease.

Zaufal (see Tilley, McBride, Parker, and others) advances the view that the disease is due to congenital defects of the intra-nasal structures, and believes that the nose is arrested in growth. He maintains that the turbinated bodies retain their infantile proportions, and seems to deny altogether the occur-

rence of atrophy. These views I cannot accept.

Charles A. Parker ("The Nose and Throat, and their Treatment", p. 307, 1906) thinks the cause of the disease is probably "an arrest of development of the turbinated bodies, caused by chronic purulent rhinitis, and increased in some individuals by hereditary peculiarities." His views seem to me a combination of those held by others. I have already mentioned the conclusion to which I have come in regard to arrest in development: anatomical peculiarities and chronic purulent rhinitis I propose to discuss later.

Greville Macdonald's theory is that the abnormal patency of the nasal cavity is due to ill-development of the maxillary antra (Greville Macdonald, A Treatise on Diseases of the Nose and its Accessory Cavities, 2nd. edition, 1892).

Parker, in criticising this theory, states, that as purulent rhinitis starts long before the maxillary antra have attained their full development, it seems likely that the causes which lead to arrested development of the inferior turbinates may also affect these accessory sinuses.

To me it seems of little consequence whether the maxillary antra are smaller or not, because it would appear that atrophy plays the chief part in enlarging the nasal cavity, this being borne out by my specimen.

In Macdonald's cases it is probably atrophy which has affected the antra during their development, being merely an extension of the atrophic changes

present in the nasal cavity. The antra are said to be affected by the disease in 18% of cases.

According to Wright, Meisser, Hopman, Kayser, Gerber, and others (see McBride, Diseases of the Throat, Nose and Ear, 1900, and Parker, The Throat and Nose, and their Treatment, 1906), the disease is determined by the racial type of skull.

I cannot admit that the racial type of skull is the primary factor in causing this disease, but I have noted it is frequently present in people with short, broad noses, and wide, upturned nostrils.

It is said, in support of this theory, that wide nasal fossae are common in people whose nasal septa are short, and whose post-nasal spaces are comparatively deep. These conditions are said to be a common accompaniment of platyrrhina or flat nose. Platyrrhina occurs in broad-faced people, who generally have the brachycephalic type of skull.

Although these facts are of interest, the disease does not occur frequently enough in such types as to warrant their being considered as causative factors. Besides, I have seen the disease in the very opposite type of skull.

I regard this theory of racial type of skull much in the same ~~way~~ light as I regard the so-called phthisical types. In a person of the phthisical type one would not be surprised to find tuberculosis developing, but this type could not be considered to be the cause of the disease; no more can one say the racial type of skull is the cause of Chronic Atrophic

Rhinitis.

In looking back at the views of the older writers, I find they confuse the disease under consideration with other conditions, such as syphilitic necrosis of the bony septum, diseases of the sinuses, tuberculous ulceration, etc.

In 1873, M. Rouge, Surgeon to the Lausanne Hospital, describes in the *Gazette Médicale de Paris*, No. 42, thirteen cases on which he operated for ozaena. In each he found what he called "some lesion of the nasal skeleton", without which, he goes on to say, the peculiar odour is not present, even in cases of ulceration or suppuration of the nasal mucous membrane. He says he removed the "bony lesions" and cured the ozaena in twelve of his cases; the thirteenth died from meningitis. He mentions how the necrosed portions may be hidden in the hollows of the turbinated bones, in the vomer or in the ethmoid; and may be concealed and kept in position by thickened and diseased mucous membrane.

At a later date, in the year 1880, we find Dr. Schaeffer of Bremen, writing on Ozaena in the *Deutsche Med. Wochenschrift*, No. 33. Ozaena, he says, has always a diathesis for its primary cause, scrofula or syphilis.

The diseases peculiar to bone and to cartilage can equally produce it, but these rarer forms ought to be distinguished from the preceding. This latter statement is no doubt an indication that he has recognised cases apart from syphilitic disease and scrofula, which were probably true atrophic rhinitis.

He describes a stage of Hypertrophy and a stage of Atrophy. In both, he says, ulcerations of the mucous membrane and deeper parts may take place. In his hypertrophic stage he describes a condition which resembles what is now known as chronic purulent rhinitis, but he confuses it with ulcerative conditions due to other causes.

In his atrophic stage he describes most accurately true Chronic Atrophic Rhinitis. He mentions all the essential features of the disease. The turbinals, he says, undergo a retrograde metamorphosis, they are atrophied; and it is this, he says, which makes the nostrils considerably enlarged.

These older views are interesting, because they shew how, in Rouge's day, true Chronic Atrophic Rhinitis was not recognised, and how later Schaeffer, from careful observations, described the essential features of the disease, although he confused it with other diseases producing ozaena. I have searched through much of the old literature on this subject, but to go further into discussions thereon would be useless, for the reason that there was no attempt at distinguishing between the various diseases which caused stench from the nostrils.

Within recent years bacteriology and special surgery have thrown much light upon diseases of the nose, clearing up many of the confused ideas which hitherto existed.

We find, on looking at other recent views on Chronic Atrophic Rhinitis, that bacteria are brought

forward as its cause. Here great difficulty exists, as authorities differ widely in their findings; further, it is impossible to shew that any one of these findings is the cause of the disease. They are, I think, the result of the disease. Again, different organisms are blamed with producing the characteristic odour. The difficulty lies in the fact that the nasal fossae normally contain organisms, especially in the region of the anterior nares, where bacteria abound.

If we look at the bacteriology of the normal nasal fossae, we find that extensive work has been done by St. Clair Thomson and Hewlett ("Micro-organisms in the Healthy Nose", M.C. Trans., Vol. LXXIII), and by Wurtz and Lermoyze. The findings of these authorities were re-investigated by Park and Wright (Watson Williams, Diseases of the Throat and Nose).

St. Clair Thomson and Prof. Hewlett, in their article on the subject, shew that at the lowest estimate 1500 organisms are inhaled into the nose every hour, while they point out that it must be a common event in the atmosphere of London for 14,000 organisms to pass into the nose in a single hour. What becomes of these organisms is an important question. It has been demonstrated that the air is practically germ-free by the time it reaches the trachea. In the vestibule the vibrissae arrest some: the secretion of the nasal mucous membrane, aided by the action of the cilia, removes them rapidly.

Wurtz and Lermoyze found that as a rule the nasal

mucus was sterile.

Thomson and Hewlett made numerous cultures from the vestibule, the vibrissae of the vestibule, and no less than 76 cultures from the interior of the nose: of the latter 64 remained absolutely sterile (84%), but numerous colonies were generally obtained by cultures from the vestibule and vibrissae.

Thomson and Hewlett do not affirm that organisms are completely absent from the Schneiderian membrane, but that under normal conditions they are quite exceptional.

They have further shewn that if a culture be deposited on the septum, the organisms are rapidly removed by the action of the ciliated epithelium, and that in two hours none of the myriads of organisms could be found by bacteriological cultures.

They further shew that, while the nasal mucus is not germicidal, it exerts an inhibitory influence on the growth of micro-organisms.

In investigating these researches, Park and Wright, making cultures from the mucus in the interior of the nose of 36 normal individuals, found that only 6 were sterile and 30 non-sterile, but in most of the latter the colonies were not numerous. Though these results demonstrate that the nasal fossae are not sterile, they shew that they are not so rich in organisms as formerly supposed. They attribute this to:-

- (1) The downflow of mucus.
- (2) Action of the Cilia.
- (3) Mucus a bad culture medium.

- (4) Filtering action of the vibrissae.
- (5) Inspired air contains few pathogenic micro-organisms.

Watson Williams mentions that it is remarkable that many children with chronic membranous rhinitis, which yields pure cultures of fully virulent diphtheria bacilli, suffer from no constitutional symptoms whatever; while the tubercle bacillus and various pathogenic cocci can rarely be found in the nasal passages of healthy individuals.

From these facts it would seem that the healthy nasal mucous membrane can deal with organisms of disease and of putrefaction. This is chiefly in virtue of the mucus it secretes, aided by ciliary action. The mucus is the essential factor, because ciliary action could not go on without the mucus to moisten the epithelial surface. Let us suppose that for some reason the mucus is scantily secreted: the mucous membrane would then become dry and covered by a viscid mucus. Particles of dust and bacteria would adhere more or less firmly to this viscid mucus. Such a viscid débris would not be all removed by blowing the nose; and in children who are too young to blow the nose or expectorate débris from the naso-pharynx there would be obstruction to respiration from accumulations. Let us further suppose that this arrest in the mucus secretion is a chronic and progressive condition. What we would ultimately expect would be decomposition of the accumulated débris. The delicate ciliated

epithelium would be destroyed; due to chronic inflammatory changes taking place on the surface of the mucosa, from the irritating products of decomposition and of bacteria.

The symptoms of such a disease would be a chronic purulent discharge from the nose -- this is the type of chronic purulent rhinitis which I maintain is the precursor of Chronic Atrophic Rhinitis. It occurs most frequently in children between the ages of six months and eight years.

Parker (The Nose and Throat and their Treatment: p.302,1906) mentions some interesting facts regarding chronic purulent rhinitis. If we wipe away the discharge in the nasal fossae, we find the mucous membrane hyperaemic, but there is no marked swelling or hyperplasia. If the fossae are kept ^{free} from secretion by wiping it away, there is no nasal obstruction. Lastly, the disease is not curable, which adds weight to the theory I am about to bring forward.

Bosworth (vide Tilley, Diseases of the Nose and Throat; p.18,1908) agrees with my view that Chronic Purulent Rhinitis develops into Chronic Atrophic Rhinitis, but I do not consider that Chronic Purulent Rhinitis is the essential cause of the latter disease as Bosworth thinks.

I look upon Chronic Purulent Rhinitis as an intermediate stage which develops in the course of the

atrophic changes which produce Chronic Atrophic Rhinitis.

Let us leave Chronic Purulent Rhinitis for the present, and turn to the causes of the Atrophy.

It is a remarkable fact that in no other form of rhinitis do we get such extensive atrophy of the parts as is present in Chronic Atrophic Rhinitis.

Coates, in his text-book of Pathology, 1903, gives the causes of Atrophy in a very comprehensive manner. Any of the following may be a cause:- (a) supply of nutrition, (b) quality of nutrition, (c) diminution in function, (d) nervous cause.

Schuchardt, Valentine, and Wingrave describe the condition of the inferior turbinate as an atrophy of the inferior turbinate bone without any evidence of bone disease (see Parker, The Nose and Throat and their Treatment, 1906). This describes exactly what I myself found. We are dealing with a true local atrophy and not a destructive bone disease.

A local atrophy, according to Greenfield and Lyon (Chapters in Pathology, 1905) may be characterised by diminution in size or an actual disappearance of the affected parts. I cannot, however, hold the view put forward by Berliner (Watson Williams, Diseases of the Nose and Throat, 1901) that Chronic Atrophic Rhinitis is due to a local "pressure Atrophy", associated with nasal obstruction and pressure of the turbinal against the septum. The atrophy in Chronic Atrophic Rhinitis is general throughout the nasal cavity and

not confined to the turbinal. Further, we never find turbinal obstruction in Chronic Purulent Rhinitis (Parker).

It cannot be looked upon as an "Atrophy due to loss of functional activity"; because it is very rare in individuals with narrow noses and adenoids who breathe through the mouth and do not use the nose.

It cannot be considered an "Atrophy due to excessive use", because in other parts of the body this is usually preceded by hypertrophy, and we do not find true hypertrophy preceding Chronic Atrophic Rhinitis. Further, the nose cannot be used excessively in the act of respiration, although it may be blown too frequently; this, however, seldom occurs in children with Chronic Purulent Rhinitis.

"Atrophy due to defective nutrition" probably plays an important part in the disease, but this is secondary to the real cause of the disease, which I consider is an Atrophy due to neurotrophic disturbance. We find Watson Williams in agreement with this view that the cause of the disease has essentially a nervous origin. He considers it is due to a "tropho-neurosis connected in some obscure manner with sexual development, or sexual involution at the menopause"; he maintains that in the great majority of cases the disease is primarily atrophic, and has been known to arise in infancy.

If we look at Infantile Paralysis (acute ^{anterior} polio-myelitis), here ~~was~~ we have a neurotrophic disease with many features closely resembling Chronic

Atrophic Rhinitis:- soft structures, as well as bone, are affected. The first to atrophy are the soft structures, then follow the bony structures -- the bones actually shorten and shew concentric atrophy. As Coates puts it - "even the bones diminish, if the condition is prolonged."

The bony Atrophy in Infantile Paralysis cannot be looked on as solely due to loss of muscular action.

According to Greenfield and Lyon, "it appears to depend upon a direct influence on nutrition." There is another point of similarity -- this disease occurs in the first three years of life (Osler, Principles and Practice of Medicine, p. 942), about the time when Chronic Purulent Rhinitis commences; but I have seen it occur in adults, just as cases of Chronic Atrophic Rhinitis occur occasionally in adults.

If Chronic Atrophic Rhinitis is of neurotrophic origin, how are the various features of the disease explained?

On pages 24 and 25 I mentioned that Chronic Purulent Rhinitis could arise out of a condition where the mucous secretion of the nose was scanty, but it is necessary that this condition of scanty secretion should be a progressive and permanent one.

I maintain that the condition is a progressive and permanent one, due to a neurotrophic lesion.

The trophic nerves of the nose being affected

in some obscure way, there follows a glandular atrophy in the mucous membrane. The goblet cells and racemose glands become scarce, hence the scanty secretion (see pages 12 and 13). The whole mucous membrane becomes thinner, atrophic, and more vulnerable, just like the condition of the skin when its nerve supply is cut off. Up to this stage we have been dealing with Chronic Purulent Rhinitis, and, if we regard it as neurotrophic in origin, then it is easy to understand why Chronic Purulent Rhinitis is an incurable disease, and might easily go on to Chronic Atrophic Rhinitis.

It seemed remarkable to me how a disease like Chronic Purulent Rhinitis should not cause swelling of the turbinals accompanied by nasal obstruction -- the turbinals are hyperaemic, but do not cause obstruction (see page 41). This, I hold, is due to atrophy of the mucous membrane having already taken place and preventing any excessive swelling of the turbinals.

I have mentioned (page 25) my belief that Chronic Purulent Rhinitis passes slowly into Chronic Atrophic Rhinitis. This would seem to be susceptible of the following explanation:- The more the glandular atrophy progresses, the firmer and dryer becomes the secretion, and crusts form because of the inspissated condition of the mucus. Even violent blowing ^{of} the nose will

not dislodge these crusts, and hence they accumulate in the nasal fossae. At the same time the bony structures, especially the turbinals and floor of the nose, become slowly atrophied and slowly removed, causing great widening of the fossae.

This widening causes a larger amount of air to enter the nose, which causes further drying of the inspissated mucus and inflammatory products. The wider the fossae become, the more difficult is it to expel débris from the nose, because the expulsive force of the air on blowing the nose is greatly diminished, it having now to pass through a larger lumen. This aids the accumulation of crusts.

The chronic irritation and inflammation which are present increase the formation of fibrous tissue in the mucous membrane and round the arteries. This assists in the atrophic process by arresting the nutrition to the parts. The sum of these changes gives us the condition of Chronic Atrophic Rhinitis.

Let us now turn to the cause of the loss of smell from which subjects afflicted with this disease suffer.

I think this can be explained by the atrophic changes reaching up to the delicate olfactory mucous membrane.

The olfactory mucous membrane (see diagram No. 6), according to Von Brunn (A. Von Brunn, Arch. f. Mike, Anat. Bd., XXXIX, S. 632), is situated on the superior turbinal,

occupying an area a little more than 1 cm. across, and a corresponding area situated upon the septum opposite. I have shewn, on page 10, the atrophied condition of the superior turbinal, which is in itself sufficient to explain the loss of smell.

As regards the ozaena or stench, Habermann, B. Frankel, and Krause regard it as due to fatty degeneration of the glands in the mucous membrane. Abel, investigating 100 cases, and Cozzolino, investigating 42 cases, found the *Bacillus Mucosus* present in every case examined. Cozzolino also found pseudo-diphtheria bacilli in 8 of his cases, the *staphylococcus aureus* and *albus* in 9 and 7 respectively, and numerous other organisms (see Watson Williams, *Diseases of the Nose and Throat*). They think the *Bacillus Mucosus* is the cause of the crusts and foetor. The cause of the foetor is possibly due to the *Bacillus Mucosus* or to a mixture of putrefactive organisms. The less the secretion of mucus and the dryer the crusts, the more pronounced appears to be the stench. This is probably due to the wide atrophic area of the nasal fossae, in which drying takes place rapidly, the products of the drying being exhaled and carrying with them the foetid smell. Whether the ozaena is due to the *B. Mucosus* or to fatty degeneration of the glands seems to matter little, if we accept the view that the essential cause is a neurotrophic lesion -- all the bacteria and crusts as secondary to the

primary cause.

In support of my belief as to the origin of the disease, I will quote the following interesting case:- that of a man, aged 60, whom I was asked to see by a medical friend. This patient had been suffering from neuralgia of the fifth cranial nerve of 20 years' standing. The condition affected the face, forehead, and front of the scalp. Its distribution was peculiar, as it followed the lines of developmental fusion. Commencing in the incisor teeth of the upper jaw, it spread to the central part of the upper lip, it then proceeded up the lateral aspects of the nose and along the supra-orbital margins: when severe, it spread to the forehead and front of the scalp.

Apart from the neuralgia, his nose began to trouble him some years ago; he had suffered from frequent colds, but now people complained of a bad smell coming from his nose. The condition had come on gradually; it did not, he thinks, commence with the neuralgia, but began slowly after the neuralgia had been well established.

The typical foetor of Chronic Atrophic Rhinitis was present when he was examined, greenish crusts were seen in the middle and inferior meatuses and on the septum. When these were removed, superficial excoriations were seen where they had been attacked. The mucous membrane was pale, dry, thin and markedly

atrophied. The inferior turbinals were small, making the nose more roomy, so that the naso-pharynx could be seen through the anterior nares.

The mucous membrane of the oral pharynx and naso-pharynx was thin and atrophic, and appeared to be tightly stretched on the bone.

After excluding diseases of the sinuses, etc., his history was carefully gone through. Eight years ago he had been operated on for chronic glaucoma of the right eye. He had no history or appearances of syphilis, tuberculosis, or any other constitutional disease, to account for his condition. Potassium iodide with mercury had no effect on the condition.

The crusts were removed and the nasal fossae irrigated with sodium bicarbonate solution which removed all debris. This relieved the stench as it usually does, and he was given an alkaline lotion to use with a syringe in order to prevent further crust formation.

This treatment did not help the neuralgia or render the mucous membrane any less atrophic, although it made it appear cleaner.

Regarding the case as of Neurotrophic origin, a form of treatment was adopted which I have never seen described before, namely the High Frequency Electrical Effluve. This was applied to each nasal fossa, over the nose, and over the distribution of the neuralgia,

for five minutes at a sitting.

It had a marked effect in preventing the neuralgia, which usually came on at about 6 p.m., and lasted into the night, preventing sleep. It also arrested an attack when it was present.

A marked change took place in the nasal mucous membrane, its atrophic condition almost entirely disappeared, the mucous membrane appeared thicker and much more vascular. On looking into the fossae one could not tell there had been a condition of Chronic Atrophic Rhinitis. The above treatment is the only treatment I intend to discuss, because it is, I think, original, and bears directly on my view that the disease is neurotrophic in nature.

In searching through the literature on the nerve supply to the nasal fossae, I find it is not very complete. It is accepted that common sensation comes from the fifth cranial nerve. A. Schäfer (in his textbook of Physiology, 1900) mentions that our information is not very full or well-assured regarding the course taken by the post-ganglionic fibres of the superior cervical ganglion on their way to the periphery.

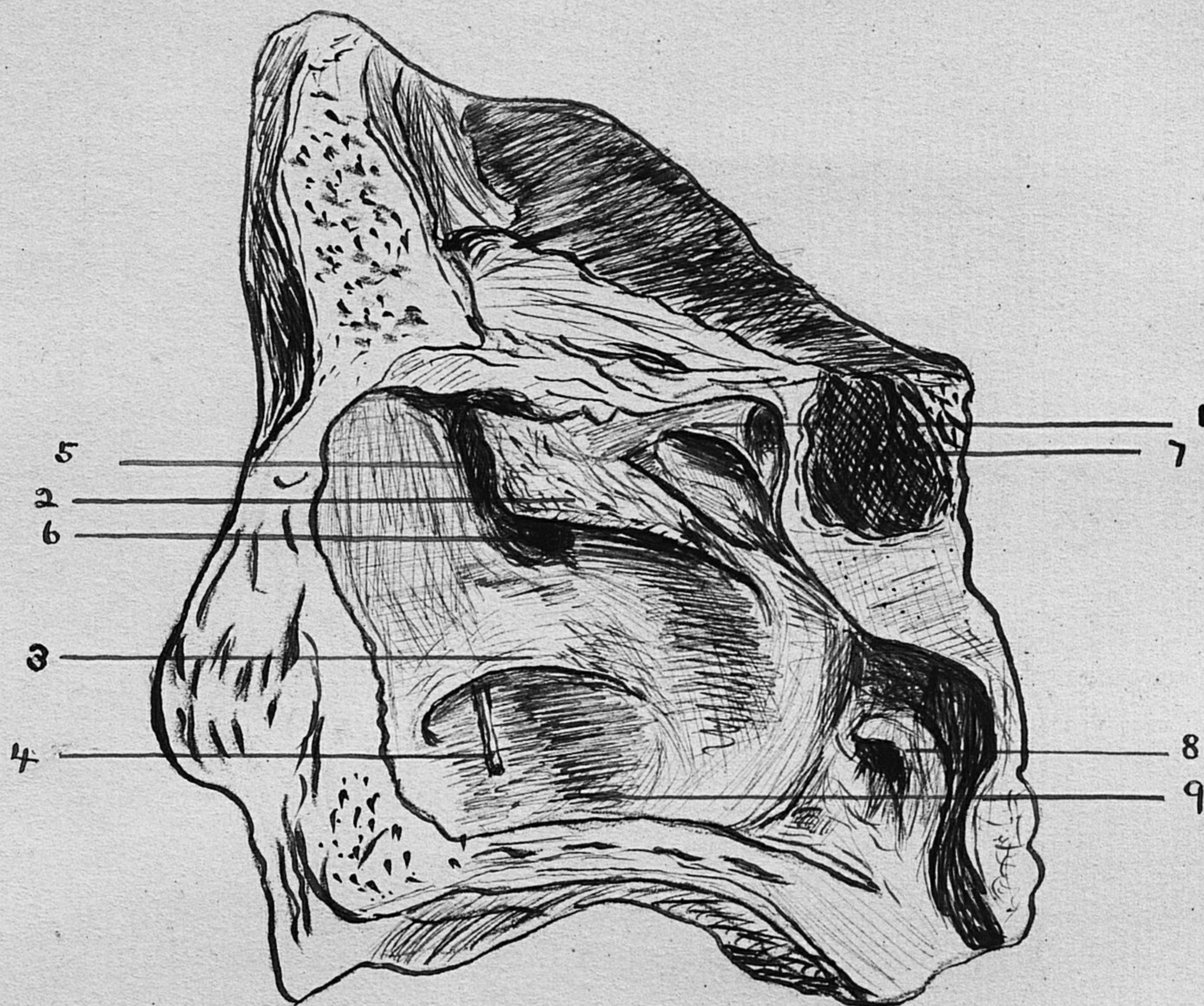
The majority of fibres which have been traced pass to the fifth cranial nerve and are distributed with its sensory fibres to the mucous membrane of the nose and mouth.

Provost (Tigerstedt's "Physiologie des Kreislaufs", Leipzig, 1893. S.480, 481) was the first to obtain evidence of naso-dilator fibres in the nasal mucous membrane on stimulation of the sphenopalatine ganglion.

Nothing appears to be known regarding the distribution of trophic nerves to the nasal mucous membrane. I conclude, however, that the primary lesion of Chronic Atrophic Rhinitis is situated in trophic nerves or their cells, and that these trophic nerves are probably distributed to the mucous membrane of the nose along with branches of the 5th cranial nerve.

The cause of the nerve lesion in Chronic Atrophic Rhinitis is as yet a mystery. Syphilis cannot be looked upon as a constant cause, as the disease occurs in those with no history of syphilis. The same may be said of Tuberculosis. Acute fevers in childhood are a possible cause source of origin; and adenoids have been put down as a cause. Probably the lesion will be found to resemble that present in infantile paralysis, the cause of which, like the causes of many nervous disorders, unfortunately still lies in obscurity.

DIAGRAM N^o 1.



KEY TO PHOTOGRAPH N^o 1

- I REMAINS OF SUPERIOR TURBINAL
- II REMAINS OF MIDDLE TURBINAL
- III REMAINS OF INFERIOR TURBINAL
- IV POSITION OF NASAL DUCT
- V THE INFUNDIBULUM
- VI SMALL UNCINATE PROCESS
- VII SPHENOIDAL AIR SINUS
- VIII ORIFICE OF EUSTACHIAN TUBE
- IX DEEP CONCAVITY NEAR NASAL FLOOR.

PHOTOGRAPH №1



CHRONIC ATROPHIC RHINITIS .

PHOTOGRAPH N^o 2.



SAGITTAL SECTION OF NASAL
CAVITY IN CHRONIC ATROPHIC
RHINITIS.

A. POSITION OF NASAL DUCT.

PHOTOGRAPH N^o 3.

SUPERIOR
TURBINAL

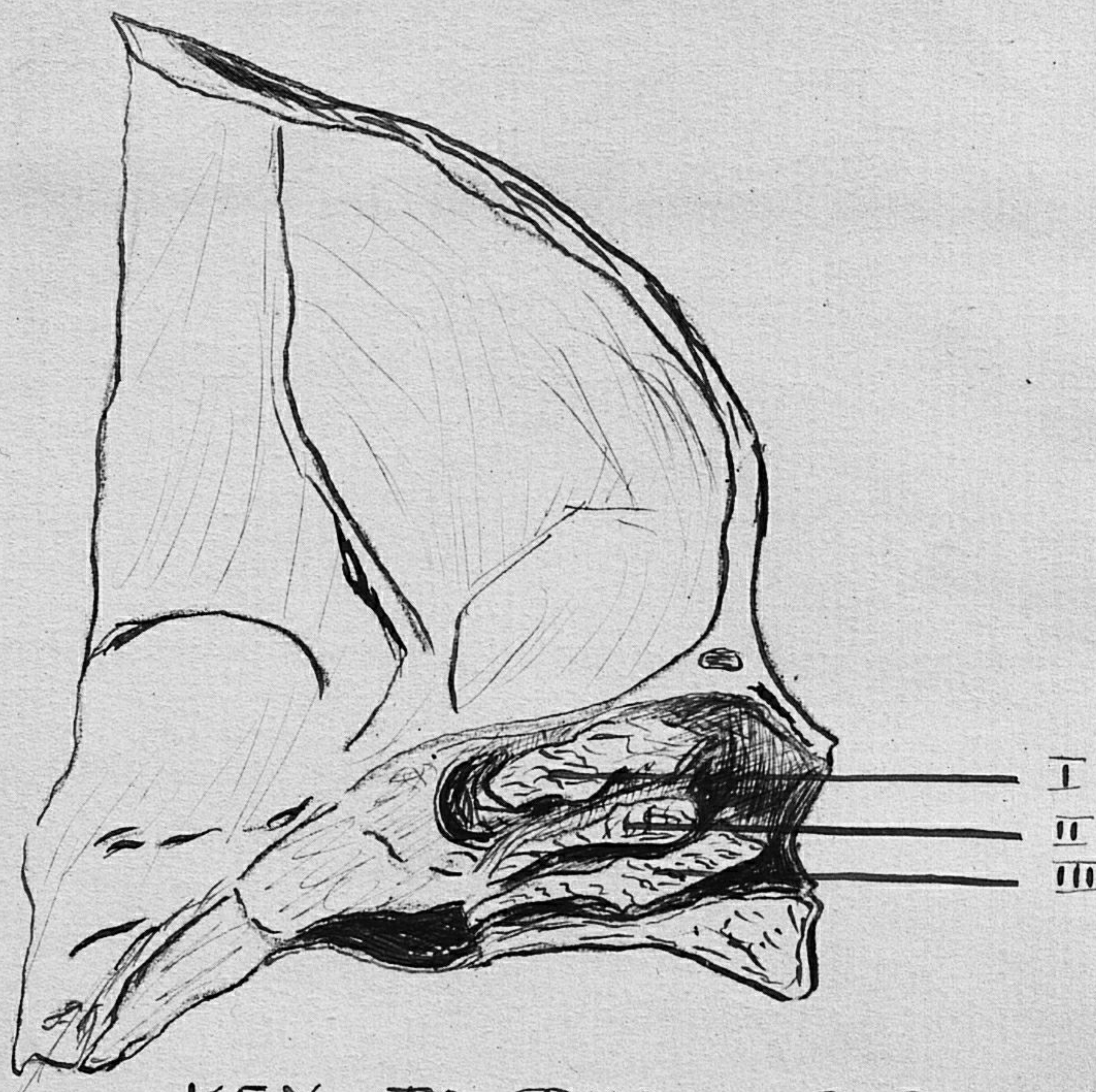


MIDDLE
TURBINAL

INFERIOR
TURBINAL

SHOWING THE THREE NORMAL TURBINALS.

DIAGRAM N^o 5



KEY TO PHOTOGRAPH N^o 4

- I SUPERIOR TURBINAL
- II MIDDLE TURBINAL
- III INFERIOR TURBINAL

PHOTOGRAPH N^o 4



SECTION OF FULL TERM FOETAL SKULL
SHEWING THE TURBINATED BONES IN
THE OUTER NASAL WALL,

PHOTOGRAPH N^o 5



SHewing THE NORMAL INFERIOR TURBINAL
FROM THREE POINTS OF VIEW.

PHOTOGRAPH N^o 6

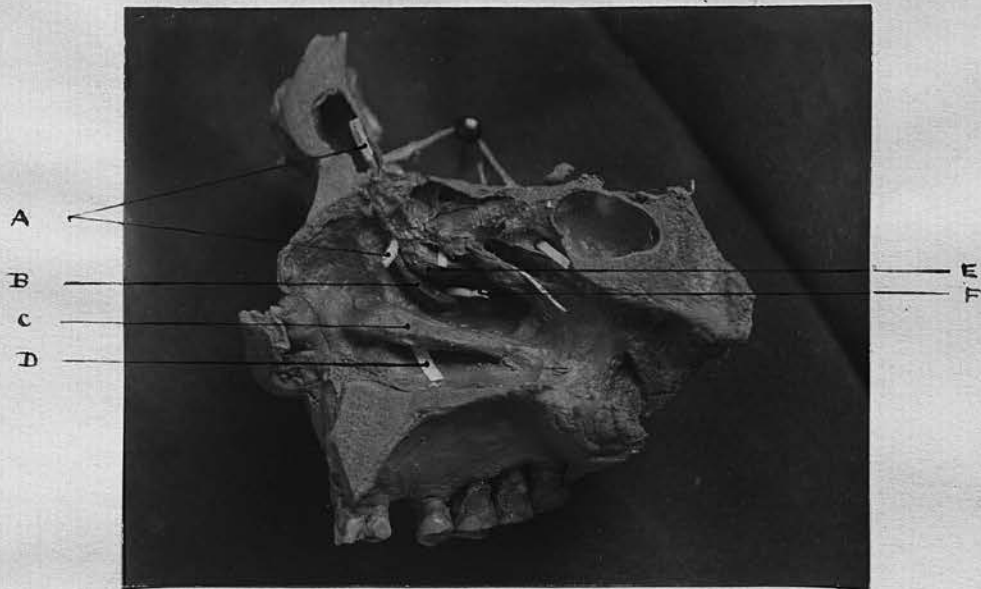
LINE OF
ATTACHMENT



INFERIOR
TURBINATED
BONE

SHEWING THE INFERIOR TURBINATED BONE DETACHED
FROM THE OUTER NASAL WALL

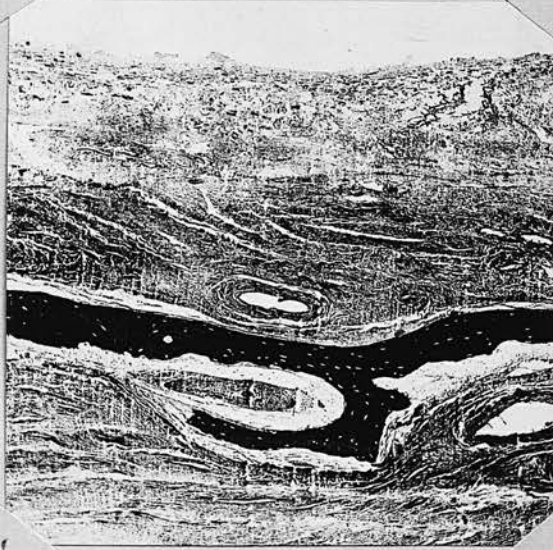
PHOTOGRAPH N^o 7



SHOWING STRUCTURES IN OUTER NASAL WALL
AFTER REMOVAL OF MIDDLE TURBINAL. [NORMAL
ANATOMY]

- A. SHOWS A WICK PASSING FROM THE FRONTAL SINUS
DOWN THE INFUNDIBULUM.
- B. THE UNCINATE PROCESS.
- C. THE INFERIOR TURBINAL.
- D. POSITION OF NASAL DUCT.
- E. ETHMOIDAL BULLA.
- F. WICK IN OSTIUM MAXILLARAE.

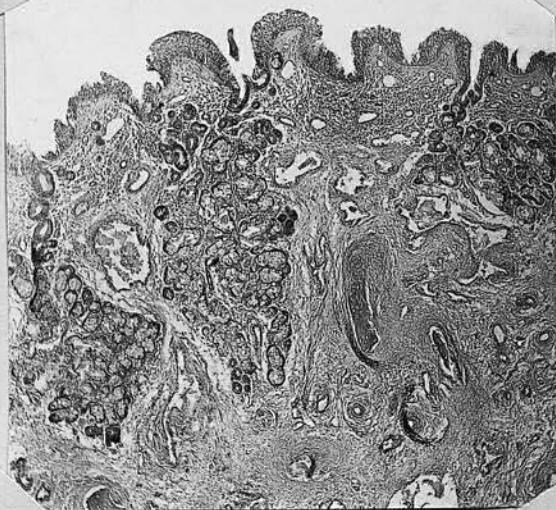
PHOTOGRAPH Nº 8.



x60. diam.

SECTION OF CHRONIC ATROPHIC MIDDLE TURBINAL

PHOTOGRAPH N^o 9.



x 40 diam.

SECTION OF NORMAL MIDDLE TURBINAL.

DIAGRAM N^o 2.

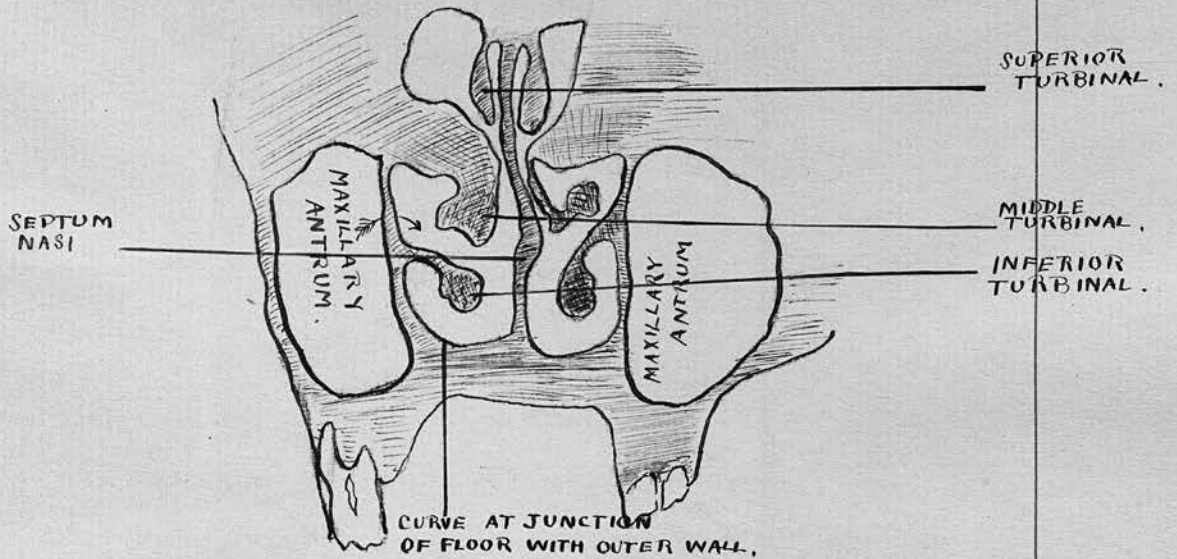


DIAGRAM N^o 3.

DIAGRAM N^o 4.

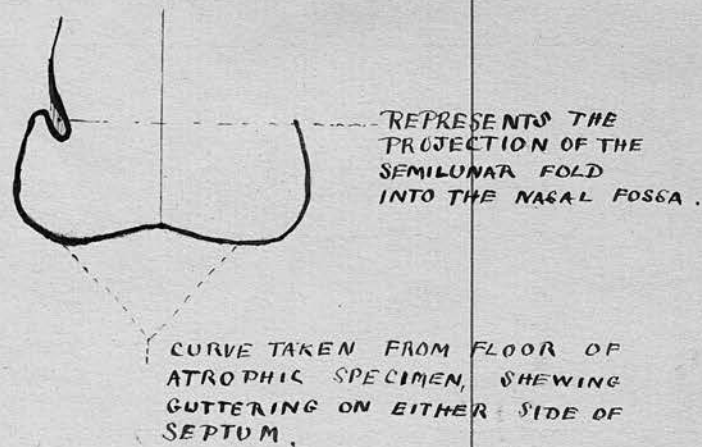
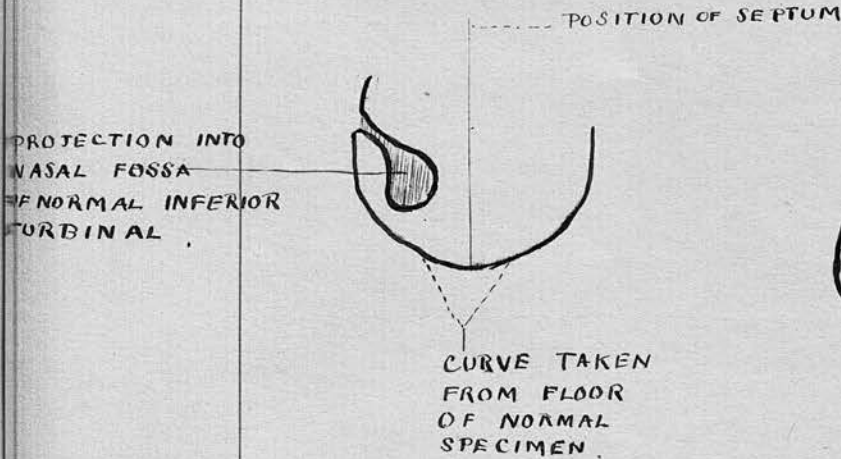
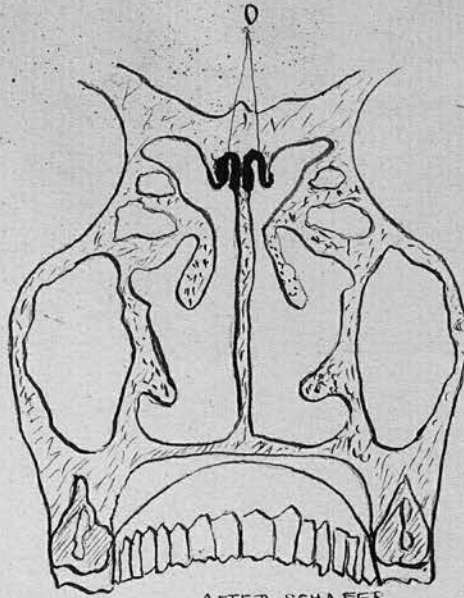


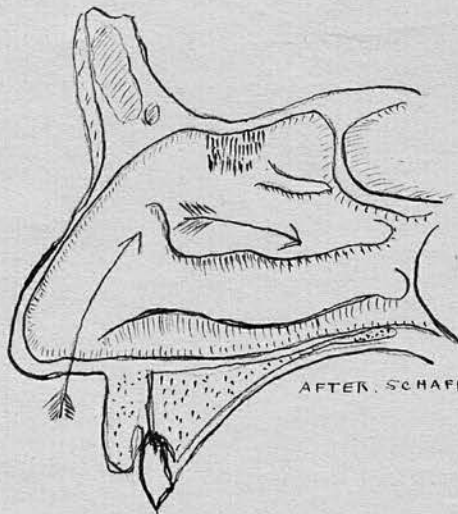
DIAGRAM. N^o 6



AFTER SCHAFER.

0. SHEWS POSITION OF OLFACTORY MUCOUS MEMBRANE OF NORMAL NOSE.

DIAGRAM N^o 7



AFTER SCHAFER.

- 'SHEWS CHIEF COURSE TAKEN BY INSPIRED AIR.